The risk ratios for passive smoking can be surprisingly high (up to 2 or 3), as reported both by Correa et al and others.[8,9] These risk ratios would be more consistent with those found for active smoking, particularly among women, if the active smoker is at greater risk also from his or her own passive smoke, again through the absorption of radioactivity on the smoke particles passively inhaled; also the relatively higher toxicity of the sidestream smoke[10] might be important.[11] These and other aspects (eg, the urban-rural difference in lung cancer risk from smoking) are more thoroughly discussed elsewhere[12] in the context of indoor radon daughters. Finally, in view of the long latency periods observed among miners acquiring lung cancer from radon daughter exposure,[13] one might suggest that the children of smoking mothers obtain an early exposure to increased levels of radon daughters at home and that smoking later in life promotes the development of lung cancer.

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LUNG CANCER AND PASSIVE SMOKING

SIR,—I was surprised to read, in Professor Trichopoulos and colleagues' letter (Sept 17, p 677), a German study of passive smoking and lung cancer described as having yielded "positive" results. The paper cited[1] contains only tentative conclusions based on poor data analysed by unacceptable methods.

I was also surprised that the findings from the Greek hospital study of passive smoking and lung cancer were almost identical to those reported two years ago[2] despite a substantial increase in the numbers of cases and controls. In the 1981 report the relative risks of lung cancer for non-smoking women were 1, 1·8, 2·4, and 3·4 according to whether their husbands did not smoke, were ex-smokers, or were current smokers of 1–20 or 21 or more cigarettes a day; the updated relative risks are 1, 1·9, 2·4, and 3·4, respectively.

In the 1981 paper the relative risks agreed exactly with the appropriate cross-product ratios calculated from the numbers of cases and controls in the relevant category for husbands' smoking. In the latest results, despite the method being apparently identical, there is a clear disagreement between the relative risks provided by Trichopoulos et al and those I calculate (see table).

RELATIVE RISK OF LUNG CANCER ACCORDING TO SMOKING HABITS OF HUSBAND

Group	Non-smokers	Ex-smokers	Cigarettes per day (current smokers)	
			1–20	21+
RR (quoted)	1·0	1·9	2·4	3·4
RR (calculated)	1·0	1·9	1·9	2·5

Relative risk = ratio of risk of lung cancer among women whose husbands belong to a particular smoking category to that among women whose husbands are non-smokers.

My calculations suggest that the latest data do not show as clear an association between a woman's lung cancer risk with her husband's smoking habits as the earlier data did. Indeed, relative risks calculated from the additional data are 1, 2·0, 1·8, and 1·8 and do not show the dose-response relation seen earlier. This doubt, added to doubts about the histological evidence and the use of cases and controls from different hospitals (limitations which Trichopoulos et al concede), prompts one to ask if the study really does add to the evidence implicating passive smoking as a factor in lung cancer.

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POTASSIUM CHLORIDE SUPPLEMENTS

SIR,—As your Round the World correspondent predicted,[1] the US Food and Drug Administration advisory committee meeting of March 2 on the controversy of wax-matrix versus microencapsulated potassium chloride preparations proved inconclusive. A few points about this controversy are worth noting.

The study by McMahon et al,[2] showing a favourable result for 'Micro-K' (A. H. Robins) in comparison with 'Slow-K' (Ciba-Geigy) was sponsored by Robins. The study by Patterson et al,[3] showing no difference between micro-K and slow-K, was sponsored by Ciba-Geigy. Both studies have been confirmed by other studies sponsored by the respective company.

Ciba-Geigy, while denying that slow-K is more ulcerogenic than micro-K, has bought from Alfred Benzon Ltd, Denmark, a licence for 'Kalinorm', a microencapsulated (pellet) preparation of KCl similar (or identical) to micro-K. It seems remarkable that Ciba-Geigy is planning to market this preparation when, according to Ciba-Geigy's US subsidiary, "Slow-K has an established clinical record unparalleled by any other solid K supplement".

It seems that, privately, Ciba-Geigy has concluded that kalinorm is as good as micro-K, and that it is better than slow-K, but they would presumably consider it scientifically incorrect to conclude that micro-K is better than slow-K.

Finally I would emphasise, as your RTW correspondent did, that doctors should "re-evaluate the decisive need for a potassium supplement and, if the indication is clear, prescribe it as a liquid". The findings of Patterson et al[3] clearly support this.

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, This letter has been shown to Dr Burley, whose reply follows.—ED. L.

SIR,—One of the main reasons why slow-release formulations of potassium were developed was the unacceptability of liquid potassium. Indeed, Patterson et al[3] reported that KCP elixir was poorly tolerated in their trial, giving rise to abdominal pain and heartburn in 9 of the 15 volunteers (60%). Dr Hansson omits to mention this. The issue is therefore whether the risk/benefit ratio of 'Slow K' is acceptable. There are eighteen years of clinical experience with slow K in the UK, during which over 4·5 million patient-years of treatment has been prescribed; with 'Micro K' formulations there is almost no clinical experience. Less than 50 cases of significant alimentary side-effects have been reported with slow K, and some of these were manifestly brought about by previous strictures or oesophageal obstruction due to cardiac enlargement. It would be hard to point to a comparable safety record with any other widely used drug. The fact that a company may be investigating or pursuing alternatives is an indication of interest and involvement in the area, and should not be interpreted as a loss of confidence in an existing product.

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